ARTICLE XII.

CASES OF LESION OF THE BASE OF THE BRAIN. I. SOFTENING OF CORPUS STRIATUM; HEMIPLEGIA NOT DEVELOPED UNTIL NEAR THE TERMINATION. II. TUMOUR INVADING OPTIC THALAMUS WITHOUT LOSS OF SENSIBILITY, BUT WITH LOSS OF CO-ORDINATION. CEREBELLAR SOFTENING.

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THE principle laid down by Hughlings Jackson, in his clinical studies, that paralysis and convulsions are not only signs of disease but supply evidence bearing upon the localization of movements and impressions in the brain and the functions of its component parts, opens a new field and offers a fresh incentive to clinical exploration. He classifies the causes of these symptoms under the now familiar titles of "discharging lesions" and "destroying lesions;" and acutely observes that "the phenomena constantly associated with such injuries, represent experiments made by disease upon the human brain, in a manner both imitating and supplementing the investigations made upon inferior animals by the hand of the physiologist."[1] We find therefore that faithful clinical observation may contribute to the progress of physiology. The fact that the functions of the cerebral ganglia of Gall, the corpora striata and optic thalami, are still unsettled, beyond the general impression, derived from their anatomical relations with the crura cerebri and the great motor tracts, that they are, in some manner, connected with motion and sensation, furnishes the motive for publishing these two interesting but isolated cases, which are offered, not in favour of any theory, but wholly with the desire to rescue from oblivion certain clinical and pathological facts, which may at some future time, and by some abler pen, be made tributary to the advance of medical science.

CASE I. *Acute White Softening of Right Corpus Striatum and Middle Lobe simulating Tumour; Hemiplegia developed towards termination of Case; Death from Coma.*—William F., a German, bookkeeper, single, æt. 36, came to me for treatment January 21, 1875. He appeared considerably older, but this was attributable to his nervous diathesis. With slight exceptions, his health had been uniformly good. He had gonorrhœa at twenty-one without sequelæ. He denied syphilis. Family history good; mother died of old age, father, aged 80, living and well. Other children healthy, except that one brother was subject to attacks of despondency bordering on melancholy, which is noteworthy in connection with the patient's tendency to become worried over trifles. In the beginning of 1872, he had a slight pharyngeal irritation and cough, and he dwelt upon this condition constantly, fearing consumption, until he became almost hypochondriacal. After a sea-voyage and spending some months among his friends, his health was greatly improved, and he continued well up to his present attack.

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[1] Clinical and Physiological Researches on the Nervous System. No. 1, On the Localization of Movements in the Brain. London, 1875.

Ordinarily temperate, a few nights before I saw him he had drunk more than usual, but not to intoxication. The night was very cold, and before retiring he bathed his face and hands in cold water. He slept heavily, and only partially covered, his left arm hanging over the side of the bed. In the morning he complained of general numbness, muscular pains and stiffness, but his left hand and forearm were completely powerless.

When he came under observation, motion had partially returned in the fingers; the paresis was more marked in the flexors of the forearm than in the extensors. Sensation was impaired on the dorsum of the thumb and index-finger, and to a less degree on the radial aspect of the hand over the first metacarpal bone. He was not prevented from attending to his business duties.

In the absence of other symptoms, the case was considered one of radial palsy due to cold and the constrained position; and treatment was instituted by hot and cold douches, friction, systematic exercise, and electricity; and, in truth under this regimen improvement at first was rapid, but soon a stage was reached where the arm continued about the same from one visit to the succeeding.

In the latter part of February some other features were noticed, which imparted a new aspect to the case, and suggested a lesion at the base of the brain. As the hand improved some rigidity appeared, which resisted flexion of the fingers and pronation and supination, but the muscles all responded well to the constant current. He complained of some thickness of the tongue, of an unaccustomed difficulty in pronouncing certain words, and of occasionally biting his tongue, which seemed too large for his mouth, although not really increased. He also called attention to numbness of the lips, and to the occasional accumulation of food on the left side of the mouth. The tongue and palatal arches showed no deviation; but an inspection of that part of his face which had been hidden by the beard, now revealed drooping of the angle of the mouth on the left side, and partial loss of expression.

On the 10th of March, the symptoms of glosso-labial palsy were less marked. The hand had nearly regained its power, and the anæsthetic area had faded away.

The next afternoon he had, for the first time, a fit. I saw him seated in a chair a few minutes later, conscious, but looking much dejected. His voice was peculiar, and a state of mental erethism was shown by his rapid ejaculatory speech and whining tone. He had been in his usual health all the forenoon; but upon rising to accompany a friend, with whom he had been chatting, to the door, he fell forward to the floor. He had vertigo; but no headache, loss of consciousness, or muscular spasm.

There was no ptosis or squint, pupils moderately dilated. He read, without hesitation, from a newspaper, when requested to do so. A watch was heard at more than two feet on the right, but at only one inch from the left ear. (Deafness had not been noticed before.) Increased left-sided facial palsy, with local anæsthesia, was observed. Tongue now protruded to the left. Forearm again hung helpless at his side. No cardiac murmur existed, the first sound was rather weak.

After taking some alcoholic stimulant, he was able to ride home, but during the night had another attack, and fell out of bed. He now sought admission to the Orthopædic Hospital, under the care of Dr. S. Weir

Mitchell, to whose courtesy I am indebted for permission to complete this case from the hospital record book.

Ophthalmoscopic examination was entirely negative. Sensation now decidedly impaired in the hand. Loss of power in forearm not complete, nor confined to parts supplied by the radial nerve. Fingers were contracted, and there was also rigidity in the forearm. Impairment of sensation existed in the left side of the face. He could readily open and shut both eyes, but closed them tightly only with effort. There was loss of lateral control of the left eye (a new symptom). Tongue was protruded to the left, but he could turn it to the right. He bit his tongue in eating, but not during sleep. The branches of the right temporal artery swollen and tortuous; vessels of left temporal region were scarcely visible. He had had continual throbbing on the right side of his head for years, connected with former attacks of neuralgia.

For the first few days after entering the hospital he was delirious and restless. The paralysis in the left arm now became associated with loss of power in the left leg, which steadily increased. A blister was applied to his left temple; iodide of potassium given in five grain doses every three hours, and a drachm of bromide at bedtime. Under this treatment he apparently was improving, on March 27th, but the next day there was absolute loss of power in the left arm and leg, and incontinence of urine; mind sluggish, sleep disturbed. Iodide was discontinued, and mercurial inunctions substituted.

A few days later, though he answered questions more readily, the hemiplegia continued the same, and ptosis of right eyelid appeared. Coma set in, and he died April 3, 1875.

An ophthalmoscopic examination two days before death failed to detect any abnormality.

Autopsy.—No emaciation. Thoracic and abdominal viscera healthy; cardiac valves not diseased. Vessels of membranes congested; longitudinal sinus contained a large firm clot.

Right hemisphere prominent and fluctuating on its superior surface, in the convolutions in front of the fissure of Rolando. Right middle cerebral artery contained a firm clot from its first bifurcation, extending about one inch.

The softening on the right side extended nearly from surface to base, partially involving parietal and almost entire anterior lobe; the right corpus striatum being disintegrated and diffluent; anterior margin of optic thalamus somewhat softened.

The clot in middle cerebral artery was attached, and fully occluded the vessel. Puncta vasculosa large, and dark coloured.

Since no record was made to the contrary, the floor of the fourth ventricle was probably in general appearance healthy. A careful microscopical report of the condition of the nuclei in the medulla oblongata would prove of interest in view of the glosso-labial symptoms (paralysis of Duchenne), and the evident involvement of the portio dura and left auditory nerve. This could not be furnished, as no specimens were allowed to be removed.

CASE II. *Glio-sarcoma in Third Ventricle encroaching upon Left Optic Thalamus; Softening of Superior Surface of the Cerebellum; neither Paralysis, Convulsions, nor Anæsthesia, but marked loss of co-ordination* *in Lower Extremities; Death from coma.*—November 15, 1877, was called to see Thomas B., æt. 45, Irish, roofer, married. Found him complaining of vertigo, headache, and a constant hissing sound in his ears, like escaping steam. He could not localize his headache, and no local explanation of the tinnitus could be discovered. There was a harsh murmur taking the place of the first sound at the mitral orifice, but the heart's action was regular, and the impulse not exaggerated. Appetite good, tongue slightly coated, bowels constipated, no albumen in the urine. Some mental dulness was thought to exist, and his memory was impaired. His health was good; "he had never been sick a day in his life," and his wife testified that he had not lost a day from sickness for seventeen years. After repeated questioning, his sister recalled that fully five winters before this attack he had fallen from a step and struck the back of his head with great violence, but he was able to walk home, and went to work as usual the next morning. He had not complained in the mean time. He was of temperate habits, and free from venereal taint.

He had been exposed to the sun, and frequently during the preceding summer complained of headache; but never had been sunstruck. In the spring of 1877 he felt as well as he ever had in his life. One day in August, after lying down, he noticed a general feeling of numbness come over him, without pain or paralysis. Following this, however, he occasionally felt dizzy. Being unable to obtain employment at this time, he did not work again until the early part of November. Vertigo now annoyed him very much, and also a peculiar noise in his ears, which gradually increased. One morning, after starting for work and going a short distance, he could not remember the way to his accustomed place of business, and was obliged to return home.

He had sought medical advice Nov. 8th, but his symptoms were not relieved. He staggered and stumbled in walking as if intoxicated. The tinnitus also became aggravated, so that, on the day before I saw him, he kept his bed. Previous to this he could walk, but was unable to maintain his balance in the dark, or with his eyes closed. He had some numbness in his legs and arms, but no actual loss of sensibility. Some myopia existed, but no changes were discovered by the ophthalmoscope, except that the disks were unusually red.

He was given iodide of potassium, increased to a drachm daily, with frequent cathartics, and blisters were applied behind the ears and at the back of the neck. He rapidly improved, and Nov. 27th was allowed to sit up. During this period he had some hallucinations, but his symptoms at this note had almost entirely disappeared. Some mental lethargy was observed, which might be habitual with him. On Dec. 7th he went out for the first time. His eyes were examined by Dr. Little at the Hospital of the Jefferson Medical College, and no evidence of disease detected; there was, on the contrary, rather less congestion of the disks. In this improved condition he had remained for more than a week, not attempting any active exertion, when the symptoms gradually reappeared. It was now thought advisable to take him to the Pennsylvania Hospital, where he was received Dec. 19th, and remained until Jan. 12, 1878, under the care of Dr. Da Costa.

It should be stated that he had used Fothergill's solution of hydrobromic acid to relieve the tinnitus. The dose was cautiously increased to a drachm three times a day; no effect being noticed, it was discontinued. Bromide of potassium was also tried, without any effect upon the symptoms, except that it seemed to increase the dulness.

While at the hospital the head-symptoms remained about the same, but his staggering became more marked and he had several falls. The tinnitus and vertigo were constant when he was sitting up, but less marked and sometimes absent when lying down. He slept heavily, and felt better when he first rose in the morning than afterwards. He had no paralysis, and answered questions intelligently, but slowly. He was taken home again by his friends.

Jan. 12. Confined in bed, and given phosphide of zinc and nux vomica, and a diet consisting largely of milk. About a week afterward, in getting into bed, losing his balance and trying to regain it, he staggered backward across the room, and fell against the opposite wall. His wife subsequently reported that whenever he stood erect he had a constantly increasing tendency to stagger backwards; until, finally, towards the middle of March, attempts at walking completely failed. When he was supported on his feet, the right leg was spasmodically thrown across the left, he could not make a step; but in the recumbent posture, neither spasm nor paralysis could be detected. His knees were unable to support his body; but he was able to accomplish any required muscular movement, and kick strongly, while lying upon his back.

In the early part of February divergent squint and diplopia appeared and became permanent. There was no prominence of the globes. He generally used his right eye, but vision was unimpaired in the left. No diminution of the area of vision appeared on rough examination in either eye. The eye-grounds were normal. The slight strabismus was attributed to spasm of the left external rectus. From the beginning of his illness he showed complete disinclination to reading, although he had previously read habitually. He had not, however, complained of his eyes.

For a short period, fluid extract of ergot was given in drachm doses, three times daily. He did not get worse under this treatment, but as he did not improve, it was decided to return to full doses of potassium iodide, which promised better results, but signally failed. Blistering partially relieved the tinnitus, but not entirely. On March 13th, a seton was introduced into the back of the neck. From this date the noises and headache entirely disappeared, and he remained the last month of his life free from pain. During the last three months he gained more than twenty pounds. He had no bed-sores.

In March it was noticed that the left leg was moved quite frequently in bed. He had also complained of some stiffness in the left arm, but as it was transitory, and he had fallen out of bed a short time before, it was attributed to contusion; at all events there was no apparent rigidity, and all the movements and tactile sense were preserved unimpaired. During the last two or three weeks he had some difficulty in swallowing; the food sometimes seemed to nearly strangle him, but whether due to spasm or paralysis was not determined. He now had involuntary alvine discharges. Previously, however, he had been unable to retain his water for any length of time, and frequently passed it in bed. He slept heavily, and occasionally had hallucinations. He could not remember how long he had been sick, and frequently asked to be allowed to get up and go to his work. On the 6th of April he was more lethargic, and the next day unconscious; cheeks and forehead flushed, the rest of the face pale, nose pinched, and eyes sunken. Temperature of body not elevated; but head warmer than usual; pupils regular, responded slowly to light; pulse compressible, accelerated, not very full; respirations twenty-four in the minute, sometimes sighing. Hypostatic congestion of both lungs existed; the heart's action was failing; the right cavities overloaded. Congestion of the lungs and brain being present, he was ordered two drops of croton oil at once, to be repeated in two hours; turpentine stupes to the chest and abdomen, and sinapisms to calves of legs.[1] A rubber cushion containing ice-water was placed under his head, and cold compresses applied. In the evening the circulation had become equalized; his breathing natural; he had no cough nor fever; the bowels had been opened freely three times. The next morning he recognized his wife, ate breakfast, and conversed with his friends. The difficulty in swallowing still continued, but no paralysis nor muscular spasm existed in the extremities; he moved all of them with equal facility. During the next few days he gradually became comatose, and died April 10th, 1878.

Autopsy made by Dr. Morris Longstreth (from whose report the following is condensed).—Heart slightly enlarged, mitral valves thickened by old deposit; lungs partly consolidated (hypostatic pneumonia). Brain firm, convolutions decidedly flattened, membranes slightly congested; deposit resembling connective tissue in interpeduncular space, where part of third nerve was included and bound down, and arachnoid was thickened and adherent. Tumour, size of walnut, found in third ventricle, moulded to the interior. Pressed surrounding structures in all directions. It was wedge-like above, broad below, extending to base of brain just above corpora albicantia, and forwards as far as anterior commissure. A caudate prolongation completely blocked up the *iter e tertio ad quartem ventriculum.* Owing to this obstruction, a large quantity of serum had been imprisoned, enormously distending the fourth and to a less extent the lateral ventricles.

The growth invading the left optic thalamus contained small hemorrhagic areas; its outline was not well defined, as it shaded off into normal tissue, penetrating and pushing the parts asunder; corpus striatum and internal capsule not implicated; right geniculate bodies unaltered, left scarcely distinguishable, and corpora quadrigemina separated and flattened; fornix softened posteriorly; ventricular walls not softened. Medulla oblongata widened, nuclei not atrophied. Left crus cerebri and optic tract flattened, not otherwise changed.

Tumour originated in gray matter of middle commissure or left optic thalamus, and grew across to the right; no other attachments existed. Superior surface covered by ventricular ependyma, and striatum zonale of left optic thalamus; it presented the characteristics of *glio-sarcoma.*

Left optic globe examined. Outline of disk irregular, not prominent; vessels obscured at border, and appeared to dip. (This may have been merely a post-mortem appearance.)

Superior surface of cerebellum showed an area of softening from median line to middle of right hemisphere, and from cortex to medullary area, the latter was not involved.

Briefly reviewing a few of the prominent points, it is noticed that in the former patient the primary symptom was the radial paralysis. Two explanations of this case suggest themselves. First, the cerebral affection preceded the palsy. There was atheroma of the smaller vessels, impaired circulation, mal-nutrition, and finally degeneration of that portion of the ascending frontal convolution containing the motor centre for the left arm (Hitzig).[1] Stasis and coagulation took place in the capillaries of this area. At the time of his fit, the clotting ran backward to the vessels at the base. Softening occurred, because the disease of the minute vessels prevented the establishment of the collateral circulation.

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[1] According to the observations of Schüler, a warm, wet compress applied to the belly or back, is immediately followed by a more or less energetic and persistent contraction of the vessels in the pia mater in trephined animals. They may thus act, as remarked by Winternitz, as do pediluvia and sinapisms to the abdomen, by diminishing the amount of the blood in the brain.—*London Practitioner*, April, 1878, p. 253.

The second hypothesis is, that the paralysis of the arm was rheumatic, and it preceded and invited the disease of the motor area, from suspension of function, a pre-existing vascular degeneration favouring this condition. The subsequent course being the same.

The first supposition appears more plausible, but the latter is by no means improbable. We know that muscles in a state of rest have their blood-supply diminished, while during action it is increased; that the secreting glands follow the same law; that the hemispheres themselves have their circulation increased by emotion and sustained attention. May not this occur in any other nervous centre when its functions are elevated? If so, then a depression of function should diminish tissue-change and blood-supply, and such a condition exists in rheumatic palsy, or after amputation. In truth, this view receives strong confirmation from the observation recently made, that after amputation of a limb the corresponding area in the brain shows atrophy to a remarkable degree.[2]

Either view is on the ground that there was disease of the small vessels, and that the condition was one of thrombosis rather than embolism. The facts that the heart was absolutely healthy, and that there were no embolic patches in the lungs or other viscera, warrant this assumption. It is also believed that the plugging preceded the acute softening, since it has been positively declared by an eminent observer to be the law of this condition.

Sufficient local cause appeared to exist for the radial palsy in the constrained position and cold, and it is not denied that this may have been entirely disconnected from the central disorder, except through the coincidence of the subsequent hemiplegia of the left side. The supposition that the rheumatic paralysis was primary, indeed offers the simplest explanation of the appearance of the anæsthetic area on the hand.

In regard to the two weeks' delay of the hemiplegia after the thrombosis, it is possible to assume either that the arterial trunk was not completely occluded until this period, or that the paralysis signalized the onset of softening.

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[1] Franck and Pitre's experiments, and the analysis of cases in M. Charcot's wards, also, serve to indicate that paralysis predominates in the upper extremity when the lesion is most marked in the anterior part of the internal capsule, as it was in this case.—*Monthly Abstract*, from *London Med. Record*, Feb. 15, 1878.

[2] Case of M. Proust, presented to French Hospital Society, Boston Med. and Surg. Journal for May 16, 1878, from Medical Examiner.

Case II. was, at first, looked upon as one of chronic basal meningitis; but this view was subsequently excluded by the course of the disease. There was no fever, although the record was kept for weeks at a time; there was neither vomiting, cramps in the limbs, nor convulsions. He never had excitement nor active delirium. The examination, it is true, revealed a small amount of thickening of arachnoid and new deposit at the base, which was probably sufficient to account for the strabismus through the involvement of the trunk of the third nerve.

The involuntary fecal discharges before the appearance of coma, pointed to a discharging lesion at the base of the brain, *i. e.*, a tumour; and it has been frequently observed that involuntary alvine evacuations occur in a certain number of cases, as the *primary symptom* of tumour at the base of the brain.

The staggering backward strikingly recalled the physiological experiments of Magendie and others, and led to a diagnosis of disease of the upper surface of the cerebellum, which was, indeed, softened, although the tumour was not beneath the tentorium, as had been anticipated. In connection with this lesion of the cerebellum it is proper to state that persistent priapism did not exist, and during the latter part of the disease there were no erections whatever. In view of the fact that direct communication has been shown by Meynert to exist between one half of the cerebellum and the opposite optic thalamus, it is perhaps significant that the crossed lesion existed in this case.

That there was no actual paralysis of sensation may be thought to be due to the fact that the optic thalamus was not sufficiently destroyed to produce entire abolition of its function. It is acknowledged, however, that a general sense of numbness existed, *but it was not confined to one side.* May not this observation, coupled with the fact that the tumour grew across in the gray commissure from the left optic nucleus to the right, warrant the view that these bodies ordinarily act as a unit, not unlike the gray matter of the spinal cord? And the experiments of Brown-Séquard upon the spinal cord have shown that a relatively greater destruction of gray matter is required to paralyze sensation than to abolish motion.

The fact that hemianæsthesia was not present is quite in conformity with the experiments of Veyssière,[1] who found that this does not occur from destruction of the optic thalamus in dogs, unless the internal capsule is also injured.

The early tinnitus aurium and vertigo, with impaired hearing, suggested

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[1] Recherches sur l'Hemianæsthesie de cause cérébrale. Paris, 1874.

Menière's disease, and it is rather unfortunate that the internal ear was not made the subject of careful study. It is thought, however, that a sufficient central cause existed for the appearance of these symptoms.

It is very noticeable that this, which, being in an otherwise healthy man, approached as nearly as possible the condition of an uncomplicated tumour of the brain, should have been accompanied by so few of the symptoms that are ordinarily considered characteristic. He had no intense, persistent headache, no vomiting, no general or peripheral paralysis, no facial palsy, and, above all, no choking of the optic disks. Tonic muscular spasm was not present to any decided extent; the affections of the eye-muscles and the œsophagus were the only motor disturbances present, if we except the loss of co-ordination in the lower extremities, which approached this condition.

The remote history of injury seems to coincide with the view expressed by Obermier that gliomata may result from injury to the skull, and he observes that the history of such an occurrence furnishes one of the points in the differential diagnosis between cancer of the brain and a gliomatous tumour.[1]

It is to be remarked that no aid in the diagnosis was furnished by the ophthalmoscope in either case, although the eyes were repeatedly examined by different observers.

ARTICLE XIII.

COLD WATER ENEMATA AS A THERAPEUTIC AGENT IN CHRONIC DIARRHŒA.

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CASE I.—On July 18, 1877, I was called to see a boy four years of age (the little son of Mr. M., residing at 204 Broome Street), where the following history was elicited:—

The first summer of the child's life, when only six months old, he was attacked with summer complaint (the ordinary entero-colitis of young children), which was treated by Dr. W., who was successful as far as the acute symptoms were concerned, but he was unable to control the diarrhœa entirely. After having treated the case about one year, and acute symptoms returning in the summer, he was discharged, and the child was put under the care of Drs. A. and T. The only result their treatment had was to put the child under the influence of opium, but the boy, being of a rachitic and very delicate appearance, always had more or less diarrhœa during their treatment, and had never learned to walk. Thus far he had not gained strength nor lost much, but later on he seemed to grow weaker and weaker, and Drs. A. and T. said he would finally die of inanition.

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[1] Article on Cerebral Tumours in Ziemssen's Encyclopædia, vol. xii. p. 263.